BEDSIDE MEDICINE FOR BEDSIDE DOCTORS

An Open Forum for brief discussions of the workaday problems of the bedside doctor. Suggestions of subjects for discussions invited.

ECLAMPSIA

ETIOLOGY

J. CARL CUMMINGS, M.D. (202 Professional Building, Glendale).—Eclampsia may be defined as a culmination of a toxemia occurring in a pregnant woman, and usually accompanied by convulsions.

Convulsions not necessarily are the accompaniment of the eclamptic state, as inhibited cases of eclampsia have occurred in which convulsions were absent; and it is possible for convulsions to occur in pregnancy parturition, or the puerperium, and they may be due to other causes than eclampsia.

An interesting sidelight on the incidence of eclampsia in Vienna was written by Dr. Walter Schiller of the Kermanner Clinic, that during the years from 1914 to 1919, or over a five-year period in which more than 35,000 cases were delivered at the Kermanner Hospital in Vienna, there was not one case of eclampsia recorded. During this five-year period the patients were without meat of any form, except soup which was prepared from human bones from the soldiers which were slain on the battlefields of the Great War. He also stated that they had many cases of eclampsia following the five-year period and many previous to the five-year period.

Pituitary as an Etiologic Factor. — Theobold states that the syndrome of eclampsia could not be explained by postpituitary hyperfunction of the posterior lobe of the hypophysis. Moreover, the hypophysis leaves unexplained the fact that the patient suffering either from the nephropathy of pregnancy, or from eclampsia, almost invariably suffers from a true retention of chlorid and constipation. . . .

At the International Congress (1913), H. Tweedy suggested that ordinary food became poisonous during pregnancy; and when in this condition it gives rise to toxemia and eclampsia. He was led to such conclusion by noting the recurrence of fits in women who had partaken of food even in small quantities. It was evident that the bad effect was not produced as a result of decomposition or irritation within the intestines, for it followed very rapidly after ingestion.

He now explains how pregnancy acts as a contributing cause: The antibodies in the blood not only guard against pathogenic bacteria, but also deal with food particles after they have entered the blood, and are thus concerned in the later processes of digestion. Badly developed or absent in the new-born, they soon become active, stimulated by an antigen richly present in colustrum and the early food. Though far in excess of normal requirements, they are not unlimited. This is proved by the sickness which may follow ingestion of some unwholesome article of food or drink, and such dissimilar complaints as migraine, epilepsy, and chronic Bright's disease, will probably be found to depend on a deficient antibody reaction.

The antibodies during pregnancy are called on to fulfill a double rôle besides their normal work; they are compelled to deal with the albumin which is constantly exuding into the maternal blood from the ovum. The presence of the ovum protein is abundantly proved by the Abderhalden test, and the effect of this toxin is noted through all the degrees of toxemia. . . .

Eclampsia viewed from the above standpoint is seen to be as certain in its etiology as any other medical complaint, for the theory is in accordance with every fact known to the practical obstetrician. . . .

It is the belief of B. C. Hirst that the origin of the toxins of eclampsia is mainly in the fetal body, and to a less extent in the placenta. He states that the adult body has enough to do to take care of the incineration, oxidation and elimination of the products of its life processes. When the waste products of the fetus and the placenta must be taken care of, it is no wonder that overburdened organs break down. This is all the more likely when a heavy protein diet and inactive and sluggish bowels increase the work.

W. Poten does not believe that the cause of eclampsia lies in the placenta, because, aside from eclampsia which occurs in the puerperium, it is hard to explain why the mother and not the child should be affected by an organ which nourishes the latter. Against the theory that a toxin is the cause of eclampsia, stands the fact that so many eclamptic patients recover with remarkable rapidity. . . .

In 1914, Young suggested that the poison was elaborated during the early disintegration of a piece of placenta whose blood supply had been cut off. . . .

Symptoms.—The writer's experience in eclampsia cases occurred between the eighth and ninth month of gestation, and the signs and symptoms of preëclamptic toxemia were recorded between the fifth and seventh month of gestation.

The symptoms preceding the toxemia are albuminuria, and usually increased systolic blood pressure, slight edema, headaches, jaundice, flatulence and epigastric pains. Later symptoms are excruciating headaches, marked edema, twitching of the muscles of the arms and legs, epigastric pains of a violent character, albuminuric retinitis, convulsions, and coma.

Increasing percentage of nonprotein nitrogen retention in the blood stream is certainly an index as to the severity of the eclampsia. . . .

The persistence of deep breathing after convulsions is a sign of persisting intoxication, and the appearance of dyspnea is a very unfavorable prognostic sign.

Constitutional Peculiarities in Eclampsia.—In eclamptic patients certain constitutional characteristics are found, namely: small stature, large pelvis, and great body weight. The majority of eclamptic patients are young, and about 50 per cent are not over twenty-five years of age. . . .

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PATHOLOGY OF ECLAMPSIA

DANIEL G. MORTON, M.D. (University of California Medical School, San Francisco).—The changes produced in the body by eclampsia are numerous. Many of them have been considered as the responsible factor in the production of the disease: first, the changes in the kidney were incriminated, later the liver necroses were considered as the etiologic factor, and more recently, the disease has been attributed to changes in the posterior portion of the pituitary gland. The truth of the matter is that the etiology still remains obscure, no proof ever having been obtained that the changes in any one organ are responsible. Gradually the conception that the pathologic findings in the various organs are *all* due to some toxic agent circulating in the blood has gained ground. At the present time, this is the most universally held view. The nature of the toxic agent we do not know, but there is increasing evidence to show that it is one or several split proteins fractions, possibly coming from breakdown of placental tissue. A discussion of the etiology of the disease appears elsewhere. I merely want to make clear here the conception which most rationally explains the pathologic findings in women dying of eclampsia, namely, that the various metabolic and structural changes observed constitute a series of events first set up by the circulation of a toxic agent in the maternal blood. All structural changes may not be directly due to this poison; many may be secondary to aberrations in metabolism and function first set up in other organs by the toxic agent. For example, the toxin may increase the permeability of capillaries, which in turn may lead to thromboses and, thus infarcted, necrotic areas, which may in turn seriously alter the function of the particular organs in which they have occurred.

Death from eclampsia may result in a variety of ways. There may be cerebral hemorrhage, or fatal hemorrhage from other organs. There may be marked cerebral edema, a "wet brain," so that the function of the vital centers is interfered with; or there may be such marked pulmonary edema that death results from asphyxia. Death may be due to marked cardiac damage in the form of thromboses and consequent necroses, or death may be essentially a uremic one because of extensive renal necroses. Thus, there is no characteristic cause of death. Generalized edema is common, as are effusions into the various body cavities. Hemorrhages and thromboses are common, may be found anywhere, as in the skin, eye, brain, heart, liver, lungs, kidneys, adrenals, etc. The blood is dark and does not clot readily. The widespread extent of hemorrhages and edema, with consequent necroses, suggests a marked increase in capillary permeability, and in the make-up of the blood itself, so that it more easily escapes from its vessels.

The most characteristic pathologic findings in women dying of eclampsia are located in the liver and kidneys, though definite alterations are usually found in the brain, heart and lungs also.

Liver.—On gross examination, the liver surface may present irregular hemorrhagic areas, giving to it a mottled appearance. On section, patches of light and dark may be seen. Microscopically, the mottled appearance is seen to be due to necrotic areas characteristically situated at the periphery of the individual lobules. These areas may contain blood cells, or may be so necrotic that structural components cannot be distinguished. In some instances, there may be a general autolysis of liver cells. The necroses were thought by Schmorl to be due to degenerative changes following thromboses of the smaller portal vessels. He, as well as many subsequent observers, such as Williams and Opie, have considered these areas of perilobular necrosis as characteristic of eclampsia. Schmorl found them in seventy-one of seventy-three autopsies, and Williams has found them in every case he has examined. The modern view is that, when present, they are characteristic, but that they are not always present. The extent of the pathologic process in the liver cannot be related to the number of convulsions which the patient had before death.

Kidney.—The kidneys are usually normal in size, anemic, may present punctate ecchymoses. The capsule is not adherent. The extent of the pathologic process varies—in some it is very marked, in others, slight. The findings are degenerative changes in the epithelial lining of tubules and of vessels. Cells are swollen, disintegrated. There is cloudy swelling and often fatty degeneration. Thromboses of glomerular vessels are common. Renal changes of some degree were found by Prutz in 361 of 368 autopsies, and by Schmorl in all but one of seventy-three autopsies. In 139 autopsies, Pollack found kidney involvement in 98 per cent. Some of the earlier observers considered the renal changes as characteristic and causative. This view has been abandoned because the lesions are often of such small extent.

Dilatation of ureters and kidney pelves, pyelitis and cystitis have often been found and, indeed, death has been attributed to the latter; but these findings are usually of secondary importance.

Brain.—Edema, areas of softening and necrosis, and thrombosis of smaller vessels are the lesions commonly found in the brain. Few are without any findings. Schmorl found thrombosis and softening in fifty-eight of sixty-five autopsies, while Prutz noted edema and hyperemia in every second or third case. Apoplexy has been described in a number of instances. To the brain changes have been ascribed the convulsions which are so commonly a part of the disease.

Heart.—The heart chambers are likely to be filled with dark, viscous blood which does not clot readily. Thrombosed vessels, necrotic areas, areas of fatty degeneration, may be found.

Lungs.—There is usually marked edema and congestion. Hemorrhage and thrombosis of the smaller vessels are common findings. Pneumonia due to aspiration of material is occasionally a prominent feature. Giant cells may be found in the lung capillaries. These cells are thought to be placental in origin, and at one time the deportation of placental elements in the maternal circulation was held responsible for the disease. However, since this has often been demonstrated in pregnant women dying of some other condition, the phenomenon has lost significance as an etiologic factor.

Eyes.—Edema, more or less diffuse retinal hemorrhage, and occasionally detachment of the retina have been described. The latter condition is thought to occur during convulsions.

Placenta.—Red infarction of the placenta of considerable extent has often been found. Indeed, it is thought by many that infarction of the placenta with the absorption in the maternal circulation of the resultant protein split products is the primary cause of the disease. However, infarction of severe degree is not infrequently found in the absence of toxemia. For these reasons it is felt by many others that infarction, like all the other lesions found, is a result, not the cause of the toxemia.

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TREATMENT OF ECLAMPSIA

EMIL J. KRAHULIK, M. D. (1680 North Vine Street, Los Angeles).—When a patient develops convulsions during pregnancy, she becomes a problem of major importance. The diagnosis of eclampsia may be readily confirmed by finding albumin in a catheterized specimen of urine, and an elevated blood pressure.

Once a convulsion has occurred the preconvulsion classification is of little importance, as the general management is the same for all groups. The treatment may be considered under three heads: (1) Sedation. (2) Reduction of toxemia. (3) Termination of the pregnancy.

Sedation.—It is absolutely imperative that the patient be in a darkened room, with a nurse constantly in attendance. Exciting stimuli must be minimized, especially noises, visitors, examinations, treatments, etc.

In the Los Angeles General Hospital, magnesium sulphate intravenously continues to give satisfactory results. Twenty cubic centimeters of a 10 per cent solution is given intravenously every hour for three doses, and thereafter every four hours, if the blood pressure continues above 150, or after each convulsion if they are occurring more frequently.

When the convulsions recur at intervals of one hour or less, the magnesium sulphate may not seem adequate, and morphin, grain one-quarter, should be given hypodermically in addition. The morphin should be repeated if another interval is shorter than one hour. Morphin will not have much, if any, effect on urinary secretion, as is sometimes feared. Concurrently with the magnesium sulphate and morphin one of the barbiturates might be given at regular intervals to promote general relaxation (sodium amytal, three grains, or nembutal, one and one-half grains, every four hours). If the patient is unconscious, a double dose may be given in a rectal suppository every six hours.

Occasionally convulsions will continue to recur in spite of sedation, and the disease seems to be out of control. Sometimes we shall find that the patient is in labor, and that will precipitate convulsions. If this exciting item is not present and the convulsions continue to recur, the situation becomes more tense. For such situations a phlebotomy is suggested. This should be postponed until one is ready to withdraw enough blood to bring the blood pressure to about 100 systolic (approximately 1,000 cubic centimeters).

Reduction of Toxemia. — Efforts to increase elimination by gastric lavage and high colonic irrigation are, at best, of doubtful value. Too often convulsions are precipitated by these procedures. Diluting the toxins with fluids and increasing the urinary output are desirable.

When the convulsions seem to be under control, if the patient is not too restless, dextrose solution may be given intravenously. If much edema is present, 300 cubic centimeters of a 25 per cent solution is given; but if the edema is not marked, a larger quantity (700 to 1,000 cubic centimeters) of a 10 per cent solution is preferred. It must be given slowly so as not to embarrass a heart already under strain. The dextrose solution should be repeated at eight-hour intervals. In the absence of blood chlorid determinations, the third flask should be combined with normal salt solution.

After consciousness has been regained, water and alkaline drinks may be offered by mouth.

Termination of Pregnancy.—The mortality rate in eclampsia has declined steadily since delivery by accouchement force was discontinued. A patient cannot stand the strain of the convulsions plus the shock of a mutilating delivery. When the patient has passed the crest of the toxemia, as evidenced by the cessation of convulsions, return of consciousness, urinary secretion, and a normal pulse rate, termination of the pregnancy may be considered. It becomes imperative when the blood pressure begins to climb, suggesting a return of the eclamptic state.

Induction of labor may be attempted by giving two ounces of castor oil. Some caution against the use of quinin in eclampsia. A hot enema is given four hours after the oil. Then, if the cervix is soft, effaced, and partly dilated, the patient may be taken to the delivery room and the bag of waters broken, allowing some of the fluid to escape. Some observers claim beneficial effects for the eclampsia, and induction will be more prompt. Rupture of the bag of waters is followed by intra-

nasal application of pitocin (one-half cubic centimeter), which is removed as soon as contractions are initiated. If the cervix is firm and not dilated, there may be some uncertainty about labor starting promptly, and it may be wiser to apply pitocin intranasally without rupturing the bag of waters.

Many prefer to induce labor by using a Voorhees bag. In a multipara, it can be inserted very easily, often without any anesthesia, and usually starts labor promptly. In a primipara with a firm cervix, bag induction is discouraged. In the primipara it will prove wiser to continue observation until the cervix has softened; but if in the meantime the blood pressure begins to rise, delivery may be effected by cesarean section under local, spinal or gas anesthesia.

Labor should be made as comfortable as possible without interfering with the dilatation of the cervix. The second stage should be terminated with forceps, as the bearing down effort might precipitate additional convulsions or a cerebral hemorrhage. Convulsions have been induced by expressing the placenta when the patient was not asleep.

Postpartum Management.—Because the convulsions have been controlled and the baby safely delivered, our duty to the patient has not been fulfilled. Considerable damage to the kidneys and other organs has taken place, and opportunity must be allowed for regeneration.

Complete physical rest must be permitted until the blood pressure has returned to normal, and the albumin has disappeared from the urine. More efficient kidney rest is procured when the patient remains on a diet of milk and fruit juices for about one week, or longer if the albumin persists. Then articles high in carbohydrate may be added, and later fruits and vegetables. The required protein will be supplied by the milk. Meats should be withheld for several months.

Prognosis for Future Pregnancies.—While convulsions almost never return twenty-four hours after delivery, we continue to keep a record of the blood pressure, as a basis for our advice concerning future pregnancies.

When the blood pressure returns to its normal limits, and when the albumin disappears from the urine within two weeks, it seems quite safe to predict that the possibility of a toxemia in another pregnancy is very remote, irrespective of the severity of the eclampsia.

When albumin persists in the urine and the blood pressure remains elevated longer than ten days or two weeks, it is almost certain that the toxemia will return in a subsequent pregnancy. The longer the period of resolution, the earlier will the next toxemia make its appearance.

If the patient had prenatal observation, and there was a long period of toxemia, we could assume that some damage was done to the kidneys, and that a toxemia will return with another pregnancy.

Some prognostic value might be placed upon a long period of coma. If the patient regains consciousness rather promptly, it might be inferred that the damage is less.

The prospects will be better for future pregnancies when ample opportunity is given for kidney repair over a long period.

The Water Supply of Vienna.—A short time ago the Vienna Health Bureau celebrated the twenty-fifth anniversary of the opening of the second alpine spring aqueduct. A Festschrift, published for the occasion, contains data on the history of Vienna's water supply. Before 1836, Vienna's fresh water came from local sources and amounted to only 1,600 cubic meters (tons) daily. In that year a new source was utilized, which provided the city with an additional 10,000 tons daily. This was supposed to have been filtered, yet in 1873 an analysis showed the water to contain "cotton fibers, bird feathers, and woolen threads." In those times there were many fatal cases of typhus and cholera. The number of deaths from typhus fluctuated between 412 in 1864 and 1,584 in 1855. The average was 845 deaths annually, or 170 for each hundred thousand inhabitants. Between 1831 and 1873 more than 20,000 persons died of cholera in Vienna. The water brought in was not pure and the quantity was insufficient (20 liters a day for each inhabitant). In addition, much water was drawn from house wells, another source of disease. In 1873 the first alpine spring aqueduct was placed in service, providing pure water. Immediately the number of typhus fatalities decreased. From 1874 to 1883 the annual average was 251 as compared with the earlier 845. During that period, 80 per cent of the houses in Vienna were connected with the new water supply. Since 1888 every building in Vienna has been supplied with pure water. The number of deaths from typhus has steadily decreased. In 1934 there were only twenty-one such fatalities; in 1918 there were 170. The average mortality from typhus for each hundred thousand inhabitants in the period 1891 to 1930 was four, or one-fortieth that of the period 1851 to 1870. Since 1873, cholera has entirely disappeared. In that year there were still 2,854 fatal cases recorded. Forty-seven imported cases were reported for the years 1892, 1893, 1910, 1914, and 1915. True smallpox is today practically unknown here. When the first aqueduct became insufficient, owing to the growth of the city, the second aqueduct was constructed in 1910 to bring Vienna water from a location 170 kilometers (100 miles) distant. The source of the water was located at 6,000 feet above sea level. The two aqueducts supply Vienna with about 380,000 tons daily, which means 200 liters for each inhabitant every twenty-four hours, although the average daily consumption is only 145 per capita. An abundant supply is thus assured. Vienna is known for the excellent quality of its water. The temperature of this water is 8 centigrade (46.4 Fahrenheit) both summer and winter. It is moderately hard and absolutely free from noxious infusions. Every building in Vienna is connected with this water supply.—*Vienna Correspondent, Journal of the American Medical Association.*

The Scotch Appetite.—Maybe there is some foundation for the current stories picturing the Scots as a bit overthrifty, penurious, stingy, close-fisted, and all that. Be that as it may, we at least know by the old records that the typical Scot of four hundred years ago was just the opposite. So the old chroniclers say. Thus wrote the English chronicler, Holinshed, in the year 1577:

"In Scotland they have given themselves (of late years to speak of) unto very ample and large diet, wherein as for some respect nature doth make equal with us, so otherwise they far exceed us in overmuch and distemperate gormandise, and so ingross their bodies that divers of them do oft become unapt to any other purpose than to spend their times in large tabling and belly cheer. Against this pampering of their carcasses doth Boethius very sharply inveigh."

At this same time in England white bread was much used in the diet of the upper classes, but the common people had to use, chiefly, rye. Some very coarse brown bread was made for the poor.